The interplay between infant mortality and subsequent reproductive behaviour: evidence for the replacement effect from historical population of Bejsce Parish, 18th-20th centuries, Poland

Tymicki, Krzysztof

Historical Social Research, Vol. 30 — 2005 — No. 3, 240-264

The Interplay between Infant Mortality and Subsequent Reproductive Behaviour. Evidence for the Replacement Effect from Historical Population of Bejsce Parish, 18th-20th centuries, Poland.

Krzysztof Tymicki[*]

Abstract: The paper examines the relationship between infant mortality and subsequent reproductive behaviour using data from a reconstitution of parish registers from Bejsce (1750-1968, Poland). It is expected that experience of infant death should increase the risk of subsequent parity transition due to the replacement effect. Multilevel event history analyses shows that the death of an infant significantly increases the risk of transition to subsequent birth and shortens the median duration of birth intervals among controlled fertility birth cohorts. The replacement effect is stronger at lower parities and when the previous child dies during the first 12 months of life.

1. Introduction

1.1 Theoretical background

The effect of infant mortality on fertility could be divided into the effect on the aggregate and the family level (Preston 1978). The aggregate level effect refers to changes in fertility as an adjustment to changes in general mortality rates, especially infant mortality rates (Galloway et al. 1998). The obvious case is the so-called fertility transition, which occurred in many historical populations. The drop in mortality rates led to a new equilibrium with lower fertility and mortality. Roughly speaking, reduction in mortality rates decreased uncertainty

[*] Address all communications to: Krzysztof Tymicki, Institute of Statistics and Demography, Warsaw School of Economics-SGH, Al. Niepodleglosci 162, 02-554 Warsaw, Poland, E-mail: ktymic@sgh.waw.pl.
An earlier version of this paper was presented at the European Population Conference, EPC 2003, 26-30 August 2003, Warsaw, Poland.

about the reproductive outcome and thus led to reduction in fertility. However, even in populations where infant mortality rates are relatively low there is always some degree of uncertainty about the survival of the newborn child. The aggregate measures of infant mortality give a crude information about the rate of occurrence of the events on the individual level, although they do not give any information about the behavioural response for such an event. This information could be provided by the investigation of the family specific effect of infant death on subsequent reproductive behaviour.

There are only sparse empirical studies that investigate the effect of infant mortality on fertility behaviour on the family level. These studies concern mostly traditional populations and provide evidence that the death of an infant had an accelerating effect on the transition to next birth (Chowdhury et al.1976; Knodel 1982; Lehrer 1984; Grummer-Strawn et al. 1998; Rajbhandary1999).

On the family specific level, the impact of child mortality on fertility understood as a positive relation between the death of the previous child and the faster transition to subsequent birth—could be divided into three main effects (Friedlander 1977):

i) Replacement effect. This category refers us to the situation when additional births are intentionally attempted with the motivation to replace the actual death of a child or children.
ii) Insurance effect (bet-hedging strategy, hoarding effect). Parents overproduce children anticipating high and varying mortality levels in order to achieve the number of surviving children that is socially or economically desired. The insurance effect might lead to "overshooting", i.e. greater number of births than desired. This "overshooting" serves as an insurance against the loss of children and the low predictability of environment (high mortality, uncertainty of economic situation).
iii) Physiological/Compensation effect. The death of the infant might trigger a physiological response (mainly through shorter breast-feeding) resulting in a more rapid return of ovulation and thus increased fecundity.

In the case of our study of the interplay between infant mortality and subsequent reproductive behaviour, physiological and compensation effects are relevant since they refer to behavioural and biological responses to the death of a child.

1.1.1 Replacement effect

When a family experiences the death of an infant it could be assumed that such an event will alter the reproductive behaviour of parents. Probably, they are going to replace the loss. As opposed to the insurance effect, which is an

answer to expected mortality, the replacement effect (as well as compensation effect) is a behavioural answer to experienced mortality (Lehrer 1984).

We have to be aware that the term replacement could mark both a type of behaviour and a demographic measure. Replacement as a demographic measure (the degree of replacement or replacement rate) stands for the average ratio of additional births to additional deaths (Preston 1978; Knodel 1982). Therefore, on the individual level replacement is complete when each lost child is replaced with a newborn infant. If the replacement is complete then a family's reaction to experienced mortality stands for a perfect compensation in terms of population rates. However, since we analyse individual level data, we are going to use the term replacement in order to describe a certain type of behaviour associated with the death of an infant.

As already noticed in Friedlander's typology (1977), the replacement effect contains a volitional component stating that parents consciously want to replace the child who just has died. Therefore it is assumed that the replacement effect will be typical for populations where family planning and deliberate fertility control are practised (Ware1977; Preston 1978). Due to the fact that in populations practicing birth control there is also a normative family size parents want to attain, in most cases the motivation to replace a dead child will be also associated with desired family size. If parents had far less children than they desired, they would reveal a stronger motivation to replace the dead infant. Thus the replacement motivation would decline as parents approach the desired family size.

Since the death of a child motivates the replacement behaviour of the parents, we may expect a significant influence of child loss both on the increased risk of parity transition and the shorter duration of the birth interval. The effect on the parity transition risk seems quite obvious—parents eagerly want to replace their loss. But why should the length of the birth interval be shorter? This could be reduced to the question, which mechanism is responsible for the duration of the birth interval. In populations where fertility control is present, couples can limit their fertility by using contraception, by postponing the formation of their union, or by reducing coital frequency. In the case of the replacement effect, especially contraceptive use and coital frequency might be of crucial importance with respect to a faster transition to the next pregnancy and birth.

1.1.2 Physiological/Compensation effect

Even in those populations where fertility control was present, replacement can not be attributed only to the volitional dimension and the parents' decision to replace their dead offspring.

It is assumed that in the populations where there is no deliberate birth control the death of the infant could also trigger a faster transition to next birth

and shorten the birth intervals. The reproductive life span in natural fertility set-up was constrained by the age at menarche (practically by the age at first marriage) and the onset of menopause. During the reproductive age the individual fecundity, i.e. the biological capacity for bearing a child, was highly determined by health status and nutrition. However, there are some physiological constraints which impose limits on the length of the birth interval (Mosley 1979).

These constraints are mainly associated with pregnancy and lactation. The fact that lactation has an inhibiting effect on the return of the ovulation is well established (Ford and Kim 1987; Leridon 1993; Le Strat and Thalabard 2001). Therefore the length of the birth interval is substantially longer in natural fertility populations practising extended breastfeeding, which is associated with longer period of so-called postpartum amenorrhea (Van Ginneken 1974; McNeilley 1993; Rodriguez and Diaz 1993). According to Bongaarts (1978), postpartum infecundability is one of the few proximate determinants that regulate fertility. Since prolonged lactation has a contraceptive effect, we may expect that sudden cessation of breastfeeding due to the death of an infant might result in a faster resumption of the ovarian cycle. This physiological mechanism is also called the reproductive compensation (Friedlander 1977; Wood 1994). Consequently, mechanisms that shorten the birth interval after a woman has lost an infant can be assumed to exist in natural fertility populations.

It is by far too simplistic to assume that both replacement and compensation mechanisms will act separately in natural and controlled fertility populations. We can rather expect some mixture of the mechanisms. For instance, in populations practicing family limitation, the physiological effects of weaning on the ovarian cycle will be also present. Nevertheless, in such populations the subsequent reproductive behaviour is consciously limited, and parents do not re-enter the natural fertility rhythm of reproduction. On the other hand, in natural fertility populations there might be some degree of birth control, which leads to the conscious motivation to replace dead offspring (Knodel 1982).

In reality it is not possible to provide a clear-cut estimation of the compensation and replacement effects. The basic distinction is one of motivation, not of behaviour: replacement is associated with the intentional substitution of a deceased child, and compensation operates behind volitional motivations.

1.2 The goals of the paper

Taking into account the theoretical considerations above, it can be hypothesised that the death of the previous infant should alter reproductive behaviour and accelerate the transition to next birth. This could be caused either by intentional replacement of deceased children or by physiological

mechanisms. The paper aims at analysing the effect of the previous child on the risk of transition to subsequent birth. Besides these basic mechanisms we want to investigate more detailed issues that might shed some light on the replacement behaviour, in particular the effect of co-variates such as parity and maternal age.

Since the data from Bejsce parish allow us to compare between natural and controlled fertility cohorts, it is possible to check the assumption that infants' deaths have differential effects on transition to subsequent birth in both fertility regimes (Rutstein and Medica 1978; Lehrer 1984). The data used are unsuitable to investigate the compensation effect directly, because there is no couple-level information on breastfeeding habits. Moreover, it seems that women did not rely strictly on breastfeeding. Anthropological evidence suggests that in Polish historical populations there was a practice of supplementing the infant's diet with other sources of food (Kolberg 1963). Investigating the compensation effect would be possible only in cases where the mother's milk was the single source of nutrition. Therefore, for the natural fertility birth cohorts we can only hypothesise that there was no replacement effect because couples' pace of childbearing was not altered by conscious choice.

Among the birth cohorts that consciously limited their fertility, we can expect a positive effect of the previous child's death on the transition to subsequent birth. Moreover the replacement effect should be highly related to the birth order of the child that passed away, because there was a certain family size that parents wanted to achieve. Thus, the willingness to replace a first or second born infant will be stronger than for instance replacing the fourth born who might be beyond the planned family size.

Accordingly we expect that in the natural fertility cohorts there will be no positive effect of infants' deaths on the transition risks to the following births. In contrast, we anticipate a significant and positive effect among the controlled fertility birth cohorts, and this effect should decline with parity reflecting the normative family size parents want to achieve.

Finally, we want to check the effect of the age at death of the previous child on the transition to next birth. There is of course a certain age limit beyond which the replacement mechanism no longer operates. It could be assumed that we can speak of the replacement effect only in cases of a child having died sufficiently early in order to have an effect on the transition risk to next birth.

2. Data

2.1 The study site

In the year 1965, the Institute of Anthropology, Polish Academy of Science, started the research program under supervision of Professor Edmund Piasecki

that aimed at collecting demographic and anthropometric data using techniques of the parish register reconstitution. For the study site, the research team has chosen Bejsce parish located in the south-central part of Poland (for details see: Piasecki 1990).

The search criteria restricted possible choices to big, rural parishes located on fertile soils, with a long and continuous settlement history and well-preserved parish registers from the 17th to the 20th century. Bejsce parish fulfilled each of these criteria and was also homogeneous with respect to the nationality and religion of its inhabitants. Furthermore it was not exposed to any dramatic depressions like wars or plague with the obvious exception of the Second World War. The entire parish (8 villages) was founded in the year 1313 and throughout all its history has relied on agricultural production. Unfortunately information on the size of the owned land was missing or incomplete and thus not included into the database.

The primary goal of the research team was to reconstruct demographic and anthropometric data on the population. Due to data collection obstacles, researchers finally decided to reconstruct only data that allowed tracing the demographic history of the whole population and particular families. The reconstitution covers the period from 1690 to 1968; incompleteness of the data restricts its practical use to the time from 1750 to1968. These data were published and described in a monograph by Piasecki (1990). Several articles are devoted to the descriptive analysis of the Bejsce database, but do not offer an extensive statistical analysis of the variables on the individual level (compare the bibliography in: Piasecki 1990).

2.2 Sample selection and preparation

The construction of the database was guided by the requirements of event history analysis. We have pursued the strategy of merging all transition parities (beginning with transition from parity 1 to parity 2) into one database. There are two reasons for the exclusion of the first parity transition. The first reason is of methodological nature. Transition to the first birth and transition to higher order parities involve a different duration. In the case of my model the basic duration is the number of months since the last birth. This basic duration could be essentially the same for all parity transitions higher than transition to first birth. The second reason is quite simple: Being interested in the effect of the death of an infant on the transition to subsequent birth we have to start with transition from parity 1 to parity 2.

The individuals in the analysis were censored in the following cases: (i) death, (ii) lost to follow-up (migration and the end of reconstitution which is the year 1968), (iii) reaching limit of the reproductive age (45 years old), (iv) lack of next parity transition, (v) birth interval longer than 72 months. In the last case it could be assumed that the birth interval lasting more than 72 months

was related to some irregularities in the reproductive functions caused probably by sterility or missed birth (Sear et al. 2003).

The multilevel analysis was designed in order to capture the differential effect of the death of infants in the cohorts experiencing natural and controlled fertility. In the Bejsce parish the onset of transition from natural to controlled fertility could be ascribed to the beginning of the 20th century. The cohorts born before this date experienced relatively high fertility with the cohort TFR around 5.5 to 6.0. The cohorts born after 1900 were characterised by significantly lower TFR ranging from 4.0 for the birth cohort 1900-1920 to 3.0 for birth cohort 1941-1960. Thus we have decided that the year 1900 will be a threshold between natural and controlled fertility, and constructed a dummy variable accordingly. The reconstruction traced the demographic history of population up to the year 1968. Right censoring thus restricts the number of women from controlled fertility birth cohorts.

2.3 Variables considered in the analysis

The basic characteristics of the variables in terms of the number of exposures and occurrences are given in Table 1. Below we give some details about the variables used in the analysis with special reference to the analysis of the replacement effect.

2.3.1 Survival status of the previous child

In the analysis of the replacement effect, the information whether the previous child has died or survived is the key variable. In addition, if we want to account for the effect of the death of the previous child on the length of the subsequent birth interval and on parity transition risks, it is crucial to include the age of the previous child at its death.

As noted by Rutstei and Medica (1978), the death of the child before its first birthday has a significant negative impact on the length of subsequent birth interval and a positive impact on the risk of transition to next parity. The effects of the death of an older child are relatively smaller, which is probably related to the length of the mean birth interval and the duration of breastfeeding. The intensity of breastfeeding is highly variable over the birth interval, This influences the absence of ovulation and duration of so-called post-partum amenorrhea (Grummer-Strawn et al. 1998). As the child is getting older the intensity of breastfeeding declines and the chances for resumption of the ovarian functions are growing. Cross-national estimates of the duration of the post-partum amenorrhea in natural fertility populations show that the length of the non-susceptible period after delivery is approximately 10 months (Van Ginneken 1974). Therefore, as the age of the previous child is approaching the length

of the mean birth interval, it is hard to disentangle between the normal resumption of the ovarian function and the compensation effect.

Table 1. Basic statistics on the number of exposures and occurrences with respect to the survival status of the previous child and the fertility regime.

	NATURAL FERTILITY		CONTROLLED FERTILITY	
	exposures	occurrences	exposures	occurrences
Baseline risk of transition to next birth (duration splines in months)				
0-18	1,782	1,036	607	279
18-27	2,491	2,366	430	404
27-36	3,825	3,727	380	351
36-54	2,578	2,475	441	396
54-72	689	625	232	187
Multiple vs. single birth				
Singleton	13,425	10,677	3,005	1,860
multiple birth	140	99	51	27
Age of mother at given transition (in years)				
14-19	284	261	105	80
19-25	3,121	2,877	1,244	905
25-30	3,579	3,189	896	561
30-35	3,092	2,611	492	256
35+	3,489	1,838	319	85
Parity transition				
1-2	2,821	2,414	1,174	845
2-3	2,400	2,085	843	537
3-4	2,081	1,766	534	256
4-5 and higher	6,263	4,511	505	249
Survival status				
Survived first 12 months	9,037	11,420	2,754	1,675
Died within first 12 months	1,739	2,145	302	212

This reasoning is valid for natural fertility populations where the deliberate control over the reproduction process is absent. As to conscious replacement, the resumption of the ovarian function determines to a lesser extent the transition to next parity. Thus, the age of the child at death should be less important in determining the birth of the next child.. However, among the controlled fertility birth cohorts the child's age at death might also play an important role since parents may want to replace the loss as soon as possible and this might be especially related to an early child loss.

In the analysis of the compensation effect we have compared the cases of children not surviving until their first birthday with those children surviving the first 12 months of life. In the hazard model the effect of infant death was represented by a time varying covariate which switches its value at the child's age at death. The effect of death of the previous child on the hazard of subsequent parity transition was introduced into the hazard model as a categorical variable, which allowed estimating so-called relative risks for selected age categories.

2.3.2 Sex of the dead offspring

The sex of the dead offspring could potentially have a differential effect on the parents' willingness to replace it. This could be related to varying costs and benefits associated with having a son or a daughter. In agricultural societies, like those of Bejsce parish, sons were more valuable than daughters and thus parents were more likely to replace deceased boys rather than girls. Daughters were more costly in terms of dowry. Having many daughters could therefore inhibit the transition to next parity.

2.3.3 Fertility regime

As explained in the previous section, all women born before 1900 were assigned to the natural fertility birth cohorts and those born after that date were assumed to control their fertility (compare Table 1). This is an important variable that allows investigating differential replacement effects with respect to the fertility regime.

2.3.4 Multiple vs. single birth

It seems necessary to control for the multiplicity of births since experiencing a twin birth significantly decreases the risk of parity transition and lengthens the birth interval. Moreover, there is no clear pattern of replacement or compensation since even if one of the twins dies, the mother is still breast-feeding. It can be noticed that the arrival of the twins constitutes a natural and unplanned instance of the insurance effect protecting parents against unexpected events such as the death of one of the infants.

2.3.5 Age of the mother at previous birth

The age of the mother at previous birth stands for an important predictor of individual fecundity and its changes over time. Moreover it can be treated as a check whether there are differences between fertility regimes. For mothers from natural fertility birth cohorts we would rather expect a constant relation

between the age and the transition to subsequent parities reflecting the fact that fecundity is relatively constant until the age of 40, when it declines. In the case of controlled fertility cohorts the influence of age on the parity transition risk should reflect the stopping aspect of the fertility limitation process resulting in a lower transition risk well before the age of 40.

2.3.6 Parity transition

The use of a variable reporting parity, such as age of mother, is aimed at controlling for the effect of time on the risk of transition to subsequent birth. In the natural fertility cohorts, the risk of parity transition should be constant over all parities, whereas among women who control their fertility the risk of transition to next birth should be negatively related to the parity number.

Furthermore, this variable allows analysing the differential effect of the death of the child with respect to parity transition. As noted in the introduction, we expect that the replacement incentive will differ in view of parity and the fertility regime. Therefore, if we intersect the fertility regime, parity transition number and the information about the survival status of the previous child we can expect to reconstruct a quite complete pattern of the replacement effect.

2.4 Limitations of the database

The characteristics of the database imply some limitations in the study of the replacement and compensation effects. Most of the studies investigating these effects included information about the duration of lactation and post-partum amenorrhea (for instance: Grummer-Strawn et al. 1998). The data coming from the reconstitution of the Bejsce parish registers did not contain such detailed information and thus we were not able to control for these physiological variables.

3. Methods

In terms of methodology, the first attempts in studying the replacement effect were focused on the narrow meaning of the replacement associated with the average ratio of additional births to additional deaths (Brass and Barret 1978; Knodel 1982). As the methodology of demographic research improved, researchers started using more sophisticated methods like the event history approach. This methodology allowed accounting for the replacement and compensation effects on the level of single events (births and deaths) as well as for their influence on transition risk and duration of the birth interval (Lehrer

1984; Grummer-Strawn et al. 1998; Rajbhandary 1999). In the present paper we are using multilevel hazard model to explain the replacement effect.

3.1 Specification of the event history model

Event history models are quite useful when we want to account for the time dependency and for the fact of censoring in the data. Also, recently produced software makes it possible to account for unobserved heterogeneity and it allows multilevel modelling in event history models (Lillard and Panis 2000).Heterogeneity with respect to individual fecundability is one of the major problems in research focused on the correlates of reproductive behaviour in traditional or historical populations with natural fertility levels. The issue of heterogeneity basically refers to underlying differences between women in the levels of their fecundability (Larsen and Vaupel 1993). Some women are more fertile due to their better health status or genetic endowment. Therefore heterogeneity might obscure true relationships between studied variables and cause severe difficulties in isolating proper causal relationships (Vaupel and Yashin 1985).

The mathematical representation of the transition rate in the multilevel model containing unobserved heterogeneity could be given by the following formula:

$$\ln \mu_{ij}(t) = y(t) + \sum_{k} \beta_{jk} x_{ijk} + \sum_{k'} \gamma_{k'} v_{jk'} + u_{ij} + \delta_i \qquad (1)$$

where μ_{ij} is the intensity and (t) stands for basic duration (here time in months since last birth). Thus the whole term $\mu_{ij}(t)$ refers to the rate of occurrence of an event at time t (the birth of j^{th} infant) for the i^{th} woman. The component y(t) captures the baseline hazard (i.e. the effect of duration on the intensity of the studied event). The x_k represent the k'^{th} time constant and time varying covariates on the child level with β as the respective regression parameter. The $\gamma_{k'}$ represents the k^{th} time constant and time varying covariate on the mother's level. Two last parameters are responsible for unobserved heterogeneity; u_{ij} refers to child level heterogeneity and δ_j refers to the mother level heterogeneity factor. It is also assumed that the latter parameter δ_j is normally distributed.

So, the mathematical formulation of the model is closely associated with the theoretical arguments made above. The model has a multilevel structure and accounts separately heterogeneity factors on the level of mothers and children. This was necessary since we have included all parity transitions of each woman into one database, which requires a hierarchical structure of the database because one woman could contribute several children to the analysis. We assume that children from a single mother share some characteristics due to gene-

tic endowment and widely understood family background. Such an assumption violates the postulate of independence between observations that is required in single-level statistical models.

As mentioned above, unobserved differences in fecundability between women are the main source of distortions in the model. Hence the mother level heterogeneity factor enables us to remove unobserved differences in fecundability between women. This allows us to overcome problems with bias due to unobserved characteristics of the women and their influence on the replacement and compensation effect (Brass and Barret 1978). In order to estimate the multilevel hazard regression model of the influence of kin variables on transition to subsequent parities we use the aML software (Lillard and Panis 2000).

3.2 Effect of infant's death on the progression to subsequent birth

We aimed at capturing the replacement effect in two ways. First, the simplest way was to calculate the Kaplan-Meier estimate (product-limit estimator) of the survivor function conditioned on the fertility regime, survival status of the previous child, and parity. The basic description of the methodology can be found in the handbook by Blossfeld and Rower (1995). The survivor function was calculated using the STATA software.

Second, the estimation of the replacement effect was modelled using the multilevel hazard model presented in the previous section. The baseline hazard denoted as y(t) in equation (1) serves as a base for the duration effect. This baseline captures the effect of duration on the intensity of the studied event. In order to capture the effect of an infant's death we have included a time varying covariate that reports whether a woman has experienced a child's death and switches its value at the time of the child's death.

The spell begins 9 months after the birth of the previous child since this is the time when a woman enters the risk of experiencing her next birth. The aML software, which was used to calculate the models, allows to construct piecewise-linear splines over the duration of the spell. Such a function is linear within specified intervals (nodes), and it changes its slopes from one interval to another. That gives a quite handy way of modelling the transition risk over the duration of the spell. We have assumed the following nodes (in months): 9, 18, 27, 36, 54, and 72. They stand for the changes in the risk of transition to subsequent birth over time since last birth. The computation of the model provides us with the slopes for each declared node.

The inclusion of a time varying covariate reporting the death of the previous child is also of importance. In order to select only those individuals whose behaviour could be an effect of a child loss we have dropped the following cases: (i) when the subsequent child was conceived before the death of the previous one and (ii) when the mother died before the demise of the previous

child. In both these instances we can not really speak of any effect of the child's death on the parents' reproductive behaviour.

As mentioned above, an important aspect of the effect of the child's death on the reproductive behaviour could be attributed to the parity specific effect. In order to capture it in both natural and controlled fertility birth cohorts we have calculated separate models for these both groups by performing an interaction between the parity and the information about the survival status of the previous child represented by the time varying covariate. By this means we were able to account for the parity specific effect of the death of the previous child on the parity transition risk separately for cohorts exhibiting natural and controlled fertility.

3.3 The effect of the child's age at death

The second multilevel hazard model was estimated in order to capture the effect of the age of a deceased infant on the risk of subsequent parity transition. As mentioned earlier, the replacement effect is assumed to operate when the child dies relatively young, usually within the first 12 months of its life. As suggested by Lillard and Panis (2000, p. 263), the replacement effect can be effectively captured by the additional duration spline. Since we assume that the death of a child causes an increase in the hazard of transition, the introduction of the time dimension to the analysis of replacement effect might indicate whether an elevated risk persists or whether it increases or declines over time. In our model we have included the child's age at its death as a categorical variable in order to increase the accuracy of the estimation . We have compared women whose offsping died after second birthday (24 months) with those women whose child died at the age 0-3, 3-6, 6-12 and 12-24 months. According to other researches the replacement effect should be strongest in the cases when the child dies up to the twelfth month of its life and decrease after that age. Additionally, this effect should be present among controlled fertility birth cohorts.

4 Results

4.1 Kaplan-Meier estimate of the survivor function

The estimates of the survivor function inform us about the proportion of women that have experienced an event at a given time (here: months since last birth). These plots can provide us with useful information about the median duration of the birth interval or—in other words—the time in months at which half of the women progress to the next birth.

Figure 1: The Kaplan-Meier estimate of the survivor function for transition to subsequent birth with respect to the survival status of the previous child. Natural fertility birth cohorts, all parities.

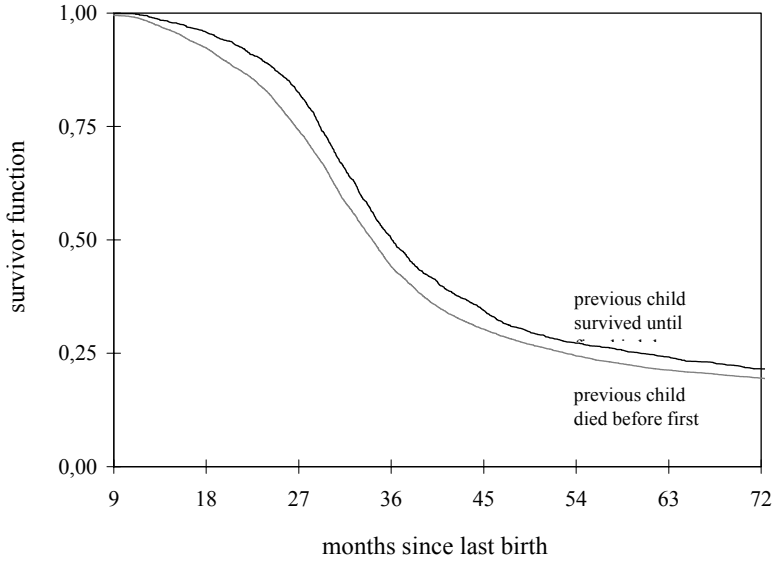
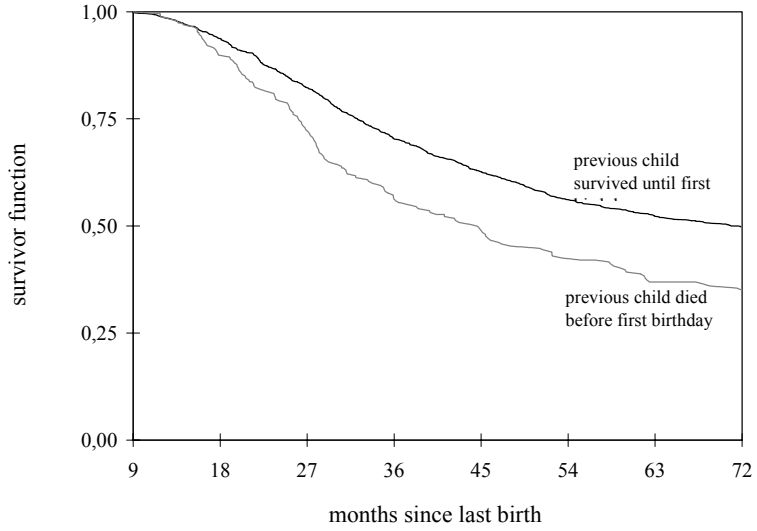


Figure 2: The Kaplan-Meier estimate of the survivor function for transition to subsequent birth with respect to survival status of the previous child. Controlled fertility birth cohorts, all parities.



In Figure 1 and 2 we have plotted the Kaplan-Meier survivor function with respect to survival status of the previous child and fertility regime. It can be noticed that there are significant differences in the pace of transition to subsequent birth between women whose child survived until first birthday and those whose child died within the first year. These differences are particularly strong among controlled fertility birth cohorts.

Table 2 summarises the duration of the median birth intervals. It can be noticed that only women from controlled fertility birth cohorts whose previous child died before first birthday experience their next birth significantly faster. There is no such effect among women from natural fertility birth cohorts (compare Figure 1 and 2). In the case of women who controlled their fertility the effect of the child's death on the median duration of subsequent birth interval is strongest at parity transition 1-2. In the other parity transitions this effect is absent or the differences are not significant.

Table 2. Duration of the median birth interval with respect to fertility regime, parity transition and survival status of the previous child based on the Kaplan-Meier estimate of the survivor function.

	NATURAL FERTILITY		CONTROLLED FERTILITY	
	Child survived until 1st birthday	Child died before 1st birthday	Child survived until 1st birthday	Child died before 1st birthday
All parities	34.14	36.06	70.82	43.80
Parity transition 1-2	31.93	32.80	59.08	30.77
Parity transition 2-3	32.83	35.49	93.16	46.77
Parity transition 3-4 and higher	35.54	38.03	75.51	114.90

It is noteworthy that there are no parity specific differences between the duration of the median birth intervals for women having experienced child loss in the natural fertility birth cohorts. Surprisingly, the differences between the median duration of the birth intervals show quite a reversed pattern: median duration of the birth interval is higher in the cases of women who did not experience the death of the previous child (however questionable the statistical significance of these differences is). This result seems to run counter the theoretical predictions. Among the natural fertility birth cohorts where only compensation/physiological mechanism is assumed to operate, the "incentive" to replace dead children should be constant across parities and show an elevated risk due to factors like cessation of breastfeeding.

Quite different patterns could be noticed among women born after the year 1900. Women who had lost their child within the first 12 months of its life progressed to next birth significantly faster than the group that did not experience such an event. The median duration of the birth interval for mothers who had lost their child is 43 months, and for the mothers of survivors it is around 70 months (irrespective of parity, compare Table 2). There are some major differences with respect to parity. In the case of parity transition 1-2 there is a strong reaction to the death of the child. Half of those mothers that have lost their first born children experienced another birth within 30 months (almost 30 months of difference!). This is a much stronger reaction to the death of the child than in the case of natural fertility birth cohorts. The strength of the replacement effect among controlled fertility birth cohorts is diminishing with parity. The reaction is also quite strong in the case of parity transition 2-3. For the parity transition 3-4 and higher the difference between median birth interval changes its direction in favour of longer waiting times for those women who have lost their child. Possibly, couples with a higher number of children were closer to their desired family size and thus not inclined to replace a dead one.

These results of the analysis of the Kaplan-Meier estimate of the survivor function provide quite a rough approximation of the replacement effect as a response to the death of a child. It will be interesting to see whether these results overlap with the estimates of the multilevel hazard model.

4.2 Multilevel hazard model of parity specific replacement effect

4.2.1 Parity specific replacement effect

The behavioural and physiological responses to the death of the child should have a differential effect with respect to parity. In order to check whether this assumption holds true we have computed the model presented in Table 3 where the parity specific transition risk was calculated in view of the survival status of the previous child. In this model the set of dummy variables representing each parity transition was interacted with the survival status of the previous child represented by the time varying covariate. By this way we have obtained a set of coefficients for each transition that refer to the relative risks conditioned on the survival status of the previous child.

The analysis of this parity specific relative risk reveals a quite interesting pattern. Among natural fertility birth cohorts, the risk of transition for women whose first born child died do not differ in a significant way from those who experienced the death of an infant. For the higher parities the death of an infant is negatively related to the relative risk of subsequent transition, although the significance is relatively weak. On average and beyond the parity transition 1-2, women whose child died have an around 18% lower risk of transition to subsequent parity. The significance of the coefficients is weak, which makes

the assumption plausible that there was no replacement effect for the women from natural fertility birth cohorts. These results seem to be quite consistent with the figures obtained from the Kaplan-Meier estimation of the survivor function.

On the other hand, among the birth cohorts exhibiting fertility control the levels of increase in the risk caused by the death of the previous child seem to vary significantly across parities. Women who lost their first child have even a four times higher risk of parity transition than the reference category. The strength of the replacement effect seems to decline with parity. Those women who lost their second or third child have only a 50% higher risk of parity transition. Once again the relations become reversed at higher parities—beyond parity transition 4-5 the death of an infant decreases the risk of transition to next birth.

The pattern of replacement behaviour among controlled fertility birth cohorts shows how strongly the replacement effect is associated with parity. Parents having lost their first child are desperate to replace it with the next one. However, those who are approaching the desired family size show much less determination in the desire to replace the loss.

4.2.2 The effect of the control variables

As mentioned in the previous sections the sex of the deceased child could potentially have an impact on the replacement. This could be traced back to the differential economic value of sons and daughters in the agricultural societies. The expected costs and benefits could be reflected for instance by the position on the marriage market (the daughters could be potentially more costly in terms of the dowry).

Our analysis revealed that significant sex-specific effect of replacement were absent among natural fertility birth cohorts. For cohorts born after the year 1900 boys were less likely to be replaced than daughters. In the case of a deceased boy the risk of transition was around 30% lower. But the estimate has to be interpreted with caution since its significance seems to be questionable.

The age of the mother at previous birth might serve as a proxy for her biological ability to conceive another child. As predicted, women's fecundity is peaking at age 20 to 25 in both cohorts and then declines with age (compare Table 3). The pace of decline in risk of the next parity transition is much faster for women from controlled fertility birth cohorts. This reflects the stopping aspect of fertility control.

In the control variables we have also included the information whether previous birth was multiple or single. Multiplicity of pregnancy should lower the risk of subsequent parity transition because unexpected overproduction of children might alter the reproductive behaviour of parents. In the current analysis there is no evidence for such an effect.

Table 3. Results of multilevel hazard model. Parity specific relative risks of transition to next birth with the information of the child's survival status represented by a time-varying covariate.

	NATURAL FERTILITY		CONTROLLED FERTILITY	
	exp(β)	s.e.	exp(β)	s.e.
PARITY SPECIFIC RELATIVE RISKS				
Transition 1-2: (ref. cat. Child survived first year of life)				
Child died within first year	1.078	0.0790	4.012 ***	0.2164
Transition 2-3: (ref. cat. Child survived first year of life)				
Child died within first year	0.791 *	0.0913	1.492 **	0.2196
Transition 3-4: (ref. cat. child survived first year of life)				
Child died within first year	0.831 *	0.0902	1.462 *	0.3504
Transition 4-5 and higher: (ref. cat. child survived first year of life)				
Child died within first year	0.844 *	0.0621	0.833 *	0.2643
CONTROL VARIABLES				
Age of mother at previous birth (ref. cat. 14-19)				
20-25	1.341 ***	0.0354	1.165 **	0.0849
26-30	1.058	0.0361	0.843 *	0.0947
31-35	0.719 ***	0.0360	0.579 ***	0.1344
36+	0.178 ***	0.0614	0.192 ***	0.2861
Multiple vs. single birth (ref. cat. singleton)				
multiple birth	1.061	0.1033	1.180	0.1999
Sex of the deceised child (ref. cat. girl)				
boy	1.070	0.0663	0.699 *	0.2172
BASELINE RISK OF TRANSITION TO NEXT BIRTH (DURATION SPLINES)				
intercept	-8.374 ***	0.1699	-9.123 ***	0.5382
0-18 months	0.219 ***	0.0106	0.261 ***	0.0332
18-27 months	0.135 ***	0.0053	0.027	0.0173
27-36 months	0.075 ***	0.0040	-0.010	0.0157
36-54 months	-0.061 ***	0.0031	-0.022 **	0.0088
54-72 months	-0.032 ***	0.0043	-0.012	0.0090
72+ months	-0.035 ***	0.0018	-0.029 ***	0.0033
Unobserved heterogeneity (δ_j)	0.543 ***	0.0209	0.516 ***	0.0608
Log-Likelihood	-59,690.7		-8,614.43	

Notes: s.e.-asymptotic standard errors; significance: '*'=10%; '**'=5%; '***'=1%; parameters refer to relative risks-exp(β) except for the baseline risk and unobserved heterogeneity parameter (δ_j).

Table 4. The results of multilevel hazard model. Baseline risks of transition (duration splines) with the effect of age at death of the previous child on the risk of transition to subsequent birth (relative risks).

	NATURAL FERTILITY		CONTROLLED FERTILITY	
	exp(β)	s.e.	exp(β)	s.e.
THE EFFECT OF AGE AT DEATH OF THE PREVIOUS CHILD (reference category child survived: relative risks)				
0-3 months	1.028	0.045	1.385 ***	0.064
3-6 months	0.847 **	0.070	3.441 ***	0.141
6-12 months	0.807 ***	0.060	3.003 ***	0.260
12-24 months	0.667 ***	0.078	1.064	0.185
CONTROL VARIABLES				
Singleton (ref.cat.)				
Multiple birth	1.087	0.106	1.125	0.158
Age of mother at given transition (ref. cat. 14-19)				
19-25	1.207 ***	0.039	1.135 *	0.067
25-30	1.091 **	0.037	1.122 *	0.068
30-35	0.803 ***	0.038	0.957	0.085
35+	0.195 ***	0.063	0.236 ***	0.157
Parity transition (ref. Cat. transition 1-2)				
2-3	0.869 ***	0.038	0.644 ***	0.064
3-4	0.778 ***	0.041	0.597 ***	0.008
4-5 and higher	0.676 ***	0.042	0.454 ***	0.093
BASELINE RISK OF TRANSITION				
Intercept	-8.089 ***	0.171	-8.379 ***	0.295
0-18 months	0.215 ***	0.011	0.236 ***	0.018
18-27 months	0.132 ***	0.005	0.062 ***	0.009
27-36 months	0.078 ***	0.004	0.004	0.009
36-54 months	-0.061 ***	0.003	-0.042 ***	0.006
54-72 months	-0.031 ***	0.004	-0.018	0.007
72 months and more	-0.035 ***	0.002	-0.028 ***	0.002
Unobserved heterogeneity (δ_j)	0.616 ***	0.023	0.793 ***	0.051
Log-Likelihood	-59,343		-18,772.6	

Notes: s.e.-asymptotic standard errors; significance: '*'=10%; '**'=5%; '***'=1%; parameters refer to relative risks-exp(β) except for the baseline risk and unobserved heterogeneity parameter (δ_j).

4.2.3 The effect of the child's age at death on the replacement behaviour

In our statistical analysis of the intensity of replacement, we have included the child's age at death as a categorical variable, which provides information on the increase in the hazard of transition associated with selected age categories. The results are presented in Table 4. For the reference category we have chosen the cases in which the children survived at least to their second birthday. As argued above the increase in the risk of transition due to death of the previous child should be highest when the child dies within the first year of its life.

When a child reaches the age of 2 years, we can not speak of the replacement effect because the relationship between the death of the previous child and the subsequent parity transition is getting weaker.

The estimates of the relative risks show that the replacement effect is strongest when the newborn dies within a year. The reproductive behaviour of those women whose child died after that year seems not to be altered by this event. Among the controlled fertility birth cohorts those women whose child died before reaching its first birthday have a more than three times higher risk of transition to subsequent birth. The pattern of elevated risk does not persist and declines with the deceased child's age at death. For the natural fertility cohorts the age-dependent patterns of replacement show a significant decrease in the risk of transition. Such a reverse pattern could be associated with the so called hoarding effect: among natural fertility cohorts or populations, overproduction of children serves as a protection against unforeseen events like increased infant mortality. Thus an early child loss might naturally adjust the reproductive outcome to the desired level: Women having lost their previous child exhibit a decreased risk of subsequent parity transition.

The model contains a new variable that controls for the parity transition. Not surprisingly women from controlled fertility cohorts exhibit a sharp decline in the risk of transition with parity. The risk of transition beyond parity 3 is almost 50% lower in comparison with the reference category that is here parity transition 1-2. The pattern of decline in the risk associated with parity is much less sharp in the case of natural fertility birth cohorts.

5. Conclusion

The present paper aimed at analysing the effect of the survival status of the previous child on the transition risk to subsequent parity. The theoretical background separates between the replacement effect and the physiological/compensation effect (Friedlander 1977; Ware 1977; Preston 1978). The replacement effect is a behavioural response to the death of the child when parents consciously limit their fertility and there is a certain family size they want to

achieve. The physiological effect, which is also called the compensation effect, refers to non-volitional response to the death of the child. In such cases the death of the child causes a sudden cessation of breastfeeding and leads to faster resumption of the ovarian function and to transition to next conception and birth (Van Ginneken 1974).

This analysis contributes to the scattered body of evidence that the death of an infant is positively related to the transition to subsequent birth. The existing evidence suggests that there is indeed a relation between the event of experiencing a child's death and faster transition to next birth and thus shorter birth interval. This evidence comes mostly from contemporary traditional populations (Chowdhury et al. 1976; Grummer-Strawn et al. 1998; Rajbhandary 1999) and to less extent from historical European populations (Knodel 1968 and 1982). In the case of studies concerning traditional populations the nature of the data allowed to control for physiological factors and thus to provide a reliable account for the physiological effect, which is impossible in studies of historical populations. However, there are some advantages of historical data since parish reconstitution data bases cover a long period of time. This allows to make a comparison between different demographic regimes.

In our analysis we were not able to control for physiological variables like duration of lactation or the duration of the post-partum amenorrhea and its influence on the duration of birth interval. We could only assume that these mechanisms were responsible for faster transition to next parity and shorter birth interval among natural fertility birth cohorts. On the other hand we were able to capture quite precisely the effect of infants' death on the reproductive behaviour among controlled fertility birth cohorts. It was possible because of the assumption that behind higher risk of transition to next parity after a death of an infant there is a mechanism of intentional replacement. Basically, mothers who had lost their child enjoyed a higher risk of transition to subsequent birth because they wanted to replace the loss. In contrast, those individuals who did not experience such an event had a lower risk of subsequent parity transition.

Using the methods of event history analysis we have found out that the death of the previous child significantly increases the risk of transition to next birth among controlled fertility cohorts and that this effect is absent among natural fertility cohorts. In the latter case there were no major differences both in the median duration of the birth interval and the parity specific risk of transition to subsequent parity. Furthermore, women having experienced a child loss have even a lower risk of transition to subsequent parity than the rest of the sample. This result is different from the estimates obtained for traditional populations exhibiting natural fertility (for instance: Chowdhury et al. 1976). It might be argued that it is not possible to provide reliable estimates of the compensation effect for historical European populations for two reasons. First, there are no databases that would allow controlling for the physiological

variables, which seems to be a prerequisite if we want to isolate the compensation effect. Second, evidence suggests that in historical Europe we can not speak of a clear-cut relation between intensity of breastfeeding and duration of post-partum amenorrhea because additional forms of infant nutrition might obscure this relation. Once again it is possible for researchers using data from traditional contemporary populations to control for the alternative sources of nutrition and thus to isolate the compensation effect.

Therefore, the only possibility for the students of historical populations is to analyse the replacement effect that appeared as a result of the demographic change and the transition from natural to controlled fertility regime. The idea of replacement is based on the assumption that parents that control their fertility want to achieve a certain family size (stopping behaviour). The event of an infant's death motivates the replacement which brings them closer to the desired family size.

In the present analysis this effect was captured in two ways, by the time varying covariate and a categorical variable that renders the effect of child's age at death on the subsequent reproductive behaviour of parents. The first multilevel hazard model presented in Table 3 was aimed at capturing the parity specific replacement effect. It shows no effect among the natural fertility cohorts and significant and strongly positive effects among controlled fertility cohorts. The pattern of replacement behaviour among controlled fertility birth cohorts shows how strongly the replacement effect is associated with parity. Parents who have lost their first child are desperate to replace it with the next one. Those who are approaching desired family size show much less determination in the desire to replace the loss.

The risk of subsequent transition for the women who lost their first-born children is around four times higher than the risk for the women whose first-borns survived. The risk declines with parity, which reflects the fact that at higher parities individuals were closer to the desired family size and thus the incentive for the replacement was lower. These results replicate (in terms of transition risk) the pattern obtained by the Kaplan-Meier estimate of the survivor function.

The absence of a similar effect among natural fertility birth cohorts is no surprise. As argued by Knodel, among natural fertility birth cohorts "there should be no evidence of deliberate attempts to replace children who have died" (Knodel 1982, p.180). However, we should be aware that even in the absence of volitional control of childbearing there might be physiological mechanisms affecting faster transition to subsequent birth, although there is no possibility to control for these mechanisms.

Among the natural fertility cohorts from Bejsce parish there is no clear and stable pattern of replacement across all parities that could be attributed to the presence of the physiological mechanisms of reproductive compensation or

replacement. There is even a slightly negative impact of the infant's death on the risk of subsequent transition.

On the other hand, among the cohorts that consciously limited their fertility this parity-specific effect is much stronger and varies across parities showing a constant pattern of decline at higher parities. This reflects the stopping behaviour and lower incentive to replace a dead child as the parents approach a desired family size. Although we were not able to control for important physiological mechanisms like duration of the lactation or post-partum sterility, the evidence on the replacement effect is quite clear-cut and shows how death of the previous child operated differently among the cohorts of a natural fertility and those practising family limitation.

The investigation of the effect of the child's age at death showed that especially early child loss (within the first 12 months) promotes higher transition rates to subsequent parities (compare Table 4). This pattern of the age effect is present among controlled fertility birth cohorts, which reflects the overall trends in replacement behaviour. An analogous effect is absent or negative among natural fertility birth cohorts where the death of the previous child has a negative effect on the hazard of subsequent parity transition. As already argued this might be related to the possibility that early child loss could be a natural adjustment for the higher than expected fertility outcomes. Moreover, poor nutritional conditions of the natural fertility birth cohorts might influence proximate determinants of reproduction leading to a lower conception probabilities for women who experienced a child loss. The death of a newborn child might also be associated with perturbations during the delivery. Such difficult and dangerous births had much more severe consequences among natural fertility birth cohorts due to poorer hygienic conditions and worst medical care.

References

BLOSSFELD, Hans-Peter and Goetz ROWER: *Techniques of Event History Modelling: New Approaches to Causal Analysis* (New Jersey: Lawrence Erlbaum Associates, 1995).

BONGAARTS, John: »A Framework for Analysing the Proximate Determinants of Fertility«, *Population and Development Review* 4 (1978), 105-132.

BRASS, William and James BARRET: »Measurement Problems in the Analysis of Linkage Between Fertility and Child Mortality«, pp. 209-233 in Samuel H. PRESTON (ed.), *The Effects of Infant and Child Mortality on Fertility* (New York: Academic Press, 1978).

CHOWDHURY, Alauddin A.K.M. et al.: »The Effect of Child Mortality Experience on Subsequent Fertility: in Pakistan and Bangladesh«, *Population Studies* 30*,* 2 (1976), 249-261.

FORD, Kathleen, and Young J. KIM: »Distribution of Postpartum Amenorrhea: Some New Evidence«, *Demography* 24, 3 (1987), 413-430.
FRIEDLANDER, Dov: »The Effect of Child Mortality on Fertility: Theoretical Framework of the Relationship«, pp. 183-204 in *IUSSP: International Population Conference* (Liege: Mexico, 1977).
GALLOWAY, Patrick R., Ronald D. LEE and Eugene A. HAMMEL: »Infant Mortality and the Fertility Transition: Macro Evidence From Europe and New Findings From Prussia«, pp. 182-226 in Mark A. MONTGOMERY and Barney COHEN (eds.), *From Death to Birth: Mortality Decline and Reproductive Change* (Washington: National Academy Press, 1998).
GRUMMER-STRAWN, Laurence M., Paul W. STUPP and Zuguo MEI: »Effect of Child's Death on Birth Spacing: A Cross-National Analysis«, pp. 39.-73 in Mark A. MONTGOMERY and Barney COHEN (eds.), *From Death to Birth: Mortality Decline and Reproductive Change* (Washington: National Academy Press, 1998).
KNODEL, John: »Infant Mortality and Fertility in Three Bavarian Villages: An Analysis of Family Histories From the 19th Century«, *Population Studies* 22, 3 (1968), 297-318.
KNODEL, John: »Child Mortality and Reproductive Behaviour in German Village Populations in the Past: A Micro-Level Analysis of the Replacement Effect«, *Population Studies* 36, 2 (1982), 177-200.
KOLBERG, Oskar: *Dzieła Wszytskie*, vol. 3: *Kujawy: Ludowa Spółdzielnia* (Wrocław: Wydawnicza, 1963).
LARSEN, Ulla and James W. VAUPEL: »Hutterite Fecundability by Age and Parity: Strategies for Frailty Modeling of Even Histories«, *Demography* 30, 1 (1993), 81-102.
LE STRAT, Y. and J. C. THALABARD: »Analysis of Postpartum Lactational Amenorrhoea in Relation to Breast-Feeding: Some Methodological and Practical Aspects«, *Journal of Biosocial Science* 33, 4 (2001), 529-549.
LEHRER, Evelyn: »The Impact of Child Mortality on Spacing by Parity: A Cox-Regression Analysis«, *Demography* 21, 3 (1984), 323-337.
LERIDON, Henri: »Fecundability and Post-Partum Sterility: An Insuperable Interaction?«, pp. 345-358 in Ronald GRAY, Henri LERIDON and Alfred SPIRA (eds.), *Biomedical and Demographic Determinants of Reproduction* (Oxford: Calderon Press, 1993).
LILLARD, Lee and Stan PANIS: *aML Multilevel Mulitprocess Statistical Software: Release 1.0* (Los Angeles/California: EconWare, 2000).
McNEILLEY, Alan Swain: »Breastfeeding and Fertility«, pp. 391-412 in Ronald GRAY, Henri LERIDON and Alfred SPIRA (eds.), *Biomedical and Demographic Determinants of Reproduction* (Oxford: Calderon Press, 1993).
MOSLEY, Henri W.: »The Effects of Nutrition on Natural Fertility«, pp. 85-105 in Henri LERIDON and Jane MENKEN (eds.), *Natural Fertility: Patterns and Determinants of Natural Fertility: Proceedings of a Seminar on Natural Fertility* (Liege: Ordina Editions, 1979).
PIASECKI, Edmund: *Ludność Parafii Bejskiej w Świetle Ksiąg Metryklanych z XVIII-XXw: Studium Demograficzne* (Warszawa: Państwowe Wydawnictwo Naukowe, 1990).
PRESTON, Samuel H.: »Introduction«, pp. 1-17 in Samuel H. PRESTON (ed.), *The Effects of Infant and Child Mortality on Fertility* (New York: Academic Press, 1978).

RAJBHANDARY, Sameer: »The Impact of Child Death on Birth Interval: A Hazard Model Analysis of Micro Data From Nepal«, pp. 1-28 in *Discussion Papers in Economics, Working Paper 99-20* (University of Colorado at Boulder, 1999).

RODRIGUEZ, G. and S. DIAZ: »Breastfeeding and the Length of Post-Partum Amenorrhea: A Hazard Model Approach«, pp. 413-427 in Ronald GRAY, Henri LERIDON and Alfred SPIRA (eds.), *Biomedical and Demographic Determinants of Reproduction* (Oxford: Calderon Press, 1993).

RUTSTEIN, Shea and Vilma MEDICA: »The Latin America Experience«, pp. 93-112 in Samuel H. PRESTON (ed.), *The Effects of Infant and Child Mortality on Fertility* (New York: Academic Press, 1978).

SEAR, Rebecca, Ruth MACE and Ian A. McGREGOR: »The Effects of Kin on Female Fertility in Rural Gambia«, *Evolution and Human Behaviour* 24 (2003), 25-42.

VAN GINNEKEN, Jeron K.: »Prolonged Breastfeeding As a Birth Spacing Method«, *Studies in Family Planning* 5, 6 (1974), 201-206.

VAUPEL, James W. and Anatoli I. YASHIN: »Heterogeneity's Ruses: Some Surprising Effects of Selection on Population Dynamics«, *The American Statistician* 39, 3 (1985), 176-185.

WARE, Helen: »The Relationship Between Infant Mortality and Fertility: Replacement and Insurance Effects«, in *IUSSP: International Population Conference* (Liege: Mexico, 1977).

WOOD, James W.: *Dynamics of Human Reproduction: Biology, Biometry, Demography* (New York: Aldine de Gruyter, 1994).